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Radiological Abnormalities in Patients who have Survived a Myocardial Infarction

Their Possible Relationship to Aneurysm Formation

BERNARD M. GRODEN AND WILSON B. JAMES

From the Departments of Medicine and Radiology, Southern General Hospital, Glasgow S.W.1

Cardiac aneurysm, defined as a localized outpouching of the cavity of a cardiac chamber with or without outward bulging of the external surface (Schlichter, Hellerstein, and Katz, 1954), may be a more frequent complication of myocardial infarction than is generally supposed. It usually involves the left ventricle and may develop either in the early stages after the infarction or after a number of years; when it appears early it may not be sharply demarcated from the main ventricular cavity. Schlichter *et al.* (1954) found aneurysms in 20 per cent of patients who had sustained a myocardial infarction. Their comprehensive review of the published reports at that time indicated that the reported incidence of aneurysm ranged from 5 per cent (Lisa and Ring, 1932) to 38 per cent (Appelbaum and Nicolson, 1935). More recently Douglas, Sferrazza, and Marici (1962) found an incidence of aneurysms of 8·7 per cent in patients dying from myocardial infarction, Abrams *et al.* (1963) an incidence of 12·4 per cent, and Dubnow, Burchell, and Titus (1965), in a review of 2293 hearts with old or recent myocardial infarction, found about 3 per cent.

All of these were post-mortem studies and most indicate that the diagnosis is rarely made in life. By comparison, clinical reports are few and usually refer to small numbers of patients.

Recently Chapman, Amad, and Cooley (1961) reported the results in 14 patients operated on under cardiac bypass. Holmes and MacFadyen (1964) described 6 diagnosed during life, one of whom was operated on successfully, and Björk (1966) has described 14 patients, diagnosed by left ventriculography and confirmed at operation. Steinberg (1966) reported 11 patients collected over a period of 27 years, in all of whom the diagnosis was confirmed by venous angiocardiography. Evidently there is a gross discrepancy between the clinical and post-mortem diagnosis in this condition.

In an attempt to assess the incidence of post-infarction cardiac aneurysm *in vivo*, we have conducted a prospective study of patients who have survived a myocardial infarction.

Patients and Methods

Seventy-five male patients who had sustained a myocardial infarction more than three months previously were examined. They were survivors of a group of patients who are being studied in greater detail for other purposes. Their ages ranged from 35 years to 76 years (mean 59 years).

The diagnosis of myocardial infarction was made on the following: a history of chest pain suggesting a cardiac origin; Q waves and/or changes in R–ST segments of the electrocardiogram suggestive of infarction; a rise of serum glutamic oxaloacetic transaminase (SGOT) above 40 units (Dade); a rise of erythrocyte sedimentation rate (Westergren). Generally all four criteria were satisfied but the presence of the first or second and at least one other was essential for the diagnosis.

Electrocardiograms were recorded on admission, on the third and seventh days after admission, weekly during the patient's stay in hospital, and 3, 6, and 12 months after the infarct.

SGOT was estimated on venous blood on the first, second, and third days after admission by the method of Reitman and Frankel (1957).

Erythrocyte sedimentation rate was estimated on sequestrinated blood (Westergren, 1921; Dacie and Lewis, 1963) on the first day of admission, repeated five to seven days later, and once a week thereafter while the patient remained in hospital.

Each patient had a postero-anterior chest *x*-ray examination before discharge from hospital. Fluoroscopy of

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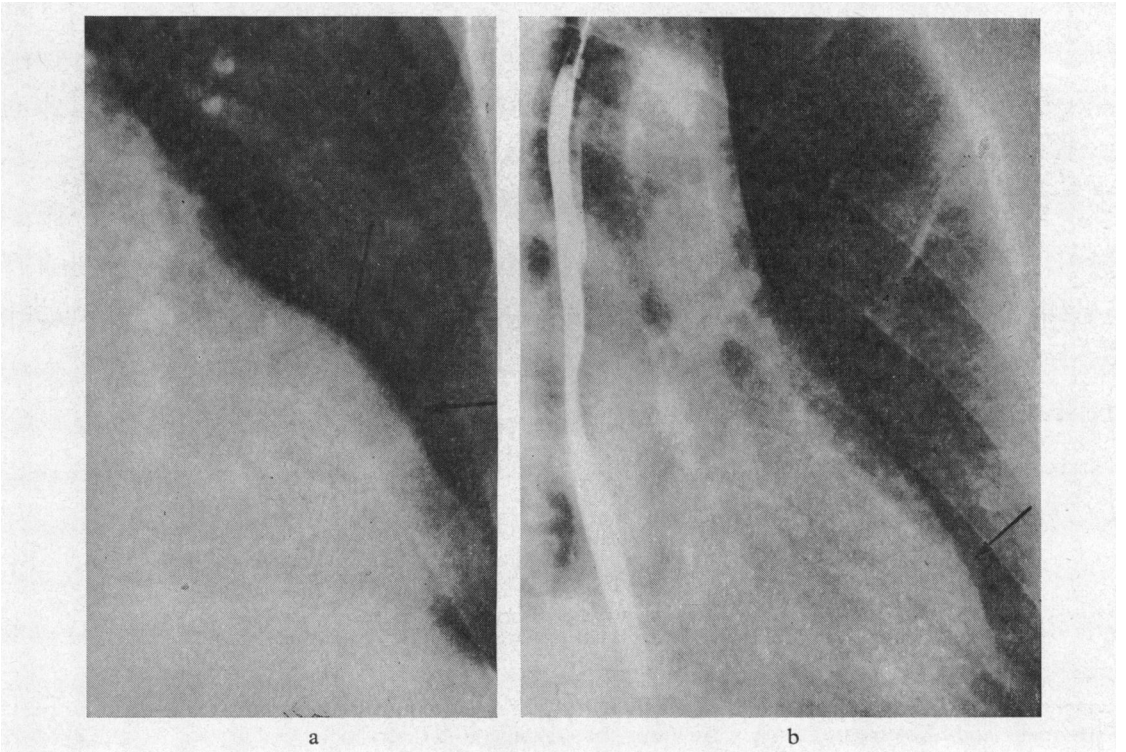

FIG. 1a.—Left heart border seen in the right anterior oblique projection, showing a large bulge above the apex (arrows).

FIG. 1b.—Right anterior oblique projection showing a small bulge above the apex (arrow).

the heart using an image intensifier television system was carried out on at least one occasion 3 to 12 months after the incident. Undercouch tube films were exposed in deep inspiration in the left lateral, right anterior oblique, and left anterior oblique projections of the heart, using a tube potential of 125 kV and without a grid, and a further postero-anterior chest radiograph was obtained at that time. Particular attention was paid to the presence of abnormal contour, pulsation, calcification, or pleuro-percardial adhesion, both during fluoroscopy and on reading the radiographs.

RESULTS

The major abnormalities detected have been grouped under the following headings: (i) bulge, (ii) abnormal pulsation, (iii) pleuro-pericardial adhesion.

Bulge. This is a localized prominence of the exterior surface of the heart (Fig. 1a and b). Bulges have been found in 15 patients, i.e. in 20 per cent of the patients examined. Some were obvious, others were minimal and seen only in tangential projection. Most bulges were associated with absent or paradoxical pulsation: 12 occurred on the anterior border of the heart, 2 on the posterior border, and 1 on the postero-inferior border. The position of 12 of these bulges corresponded to the electrocardiographic site of the infarct, and Q waves indicating transmural infarction were present on the cardiogram in 7 patients. Five patients showed R–ST elevation more than 6 months after the infarct.

Three of the patients in this group have now died. Two died suddenly at home and necropsy was not carried out. The other had a further infarction and died suddenly in hospital. At necropsy an aneurysm was discovered corresponding to the one detected radiologically (Figs. 1a, 2a and b).

Abnormal Pulsation. This is an area of diminished pulsation or one of frankly paradoxical movement when compared with the adjacent heart muscle.

We did not have access to cineradiography so that this assessment had to be made at fluoroscopy. It is admitted that this observation is a subjective one, but it is likely that abnormal pulsations occur more

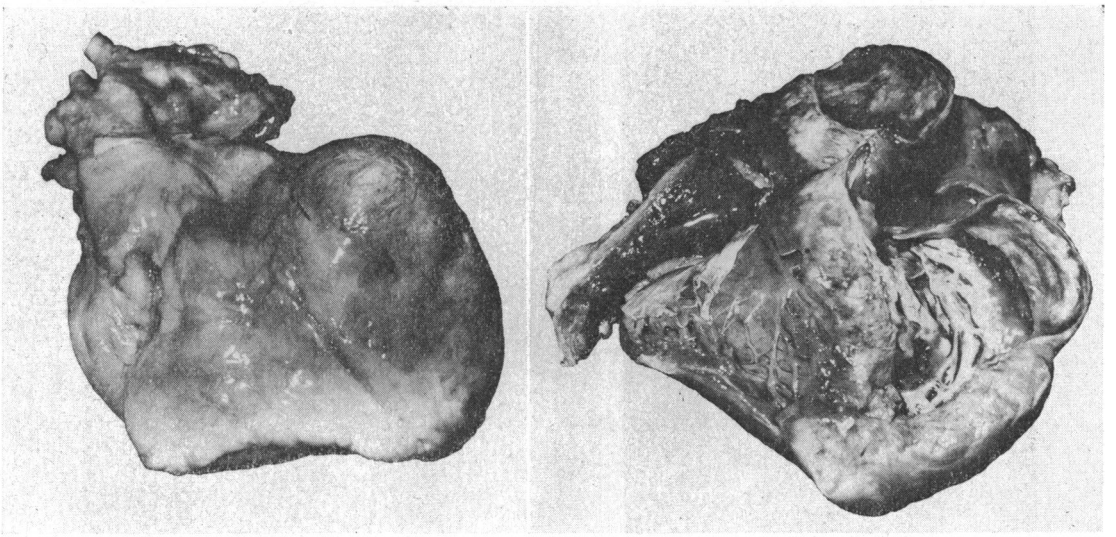

FIG. 2a.—Heart showing left ventricular aneurysm. Same patient as in Fig. 1a.
FIG. 2b.—Cut specimen showing laminated thrombus in the cavity of aneurysm.

frequently than we have been able to record (Kurtzman and Lofstrom, 1963). An area of abnormal pulsation was seen in 5 patients in our group, 4 on the anterior heart border and the other at the apex of the heart. They corresponded to the electrocardiographic site of the infarct in 4 patients.

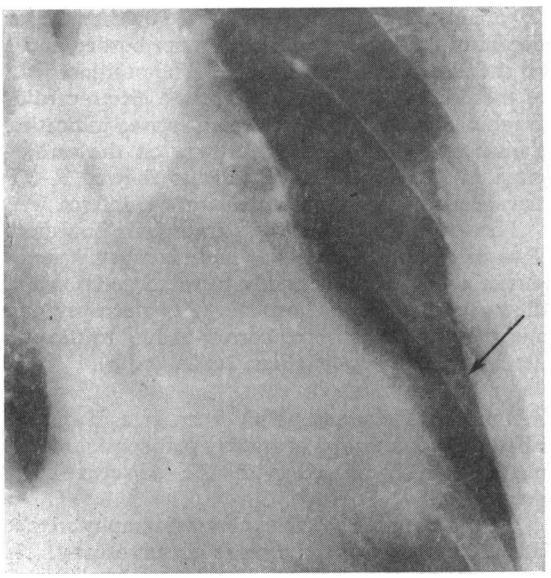

FIG. 3.—Right anterior oblique projection. Pleuro-pericardial adhesion at the apex (arrow).

Q waves occurred in one patient who also had persisting R–ST elevation.

One patient has died at home and there was no necropsy.

Pleuro-pericardial Adhesion. This is a tent-shaped opacity with its base contiguous with the heart shadow.

They are easy to demonstrate and to record on film. Their significance is debatable but there is no doubt that transmural infarction gives rise to pericarditis, and that it is likely that the end result of this process is demonstrable radiologically. Such adhesions may mask a cardiac bulge (Fig. 3).

Pleuro-pericardial adhesion was seen in 11 patients and corresponded to the electrocardiographic site of the infarct in 8. All of these occurred on the anterior surface of the heart. Q waves occurred in 6 and ST elevation persisted more than six months after the infarct in 4 patients.

Other Abnormalities. We were able to demonstrate chamber enlargement (left ventricular) in 9, coronary artery calcification in 8, pulmonary congestion due to heart failure in 4, and areas of pulmonary infarction in 2 patients.

A high incidence of cardiac enlargement is expected in patients who have sustained a myocardial infarction (Master *et al.*, 1954), and Holmes and MacFadyen (1964) feel that aneurysm should be suspected in such patients. There was nothing to

suggest in our cases, electrocardiographically or radiologically, that aneurysm formation had occurred. The results are summarized in the Table.

TABLE

RADIOLOGICAL ABNORMALITIES

Abnormality	No. of Cases
Bulge	15
Abnormal pulsation	5
Pleuro-pericardial adhesion	11
Coronary artery calcification	8
Left ventricular enlargement	9
Left heart failure	4
Pulmonary infarction	2
No abnormality	21
Total	75

Discussion

In this series, 27 per cent of patients examined showed at fluoroscopy abnormalities of cardiac outline or pulsation suggestive of aneurysm formation.

The value of contrast radiography in the diagnosis of aneurysm following myocardial infarction has been discussed recently by Holmes and MacFadyen (1964), Björk (1966), and Steinberg (1966). In many of the cases which they report, however, the diagnosis had been suspected by simple radiological methods. A diagnosis might have been made in some of our patients by left heart catheterization and ventriculography. We did not proceed with these investigations in view of the slight risk associated with them, especially in the post infarct case. We doubt whether angiography would have been helpful in the patient who died and on whom a necropsy was obtained, since the aneurysm was filled by thick adherent thrombus which presented a smooth surface to the cavity of the ventricle (Fig. 2b). It has not been established that the prognosis is worse for patients after myocardial infarction if an aneurysm develops. It is, therefore, unreasonable to subject a patient to a potentially dangerous procedure unless a diagnosis is essential as a prelude to operation. It is in cases which might be considered for operation that the diagnosis is likely to be obvious on conventional radiological examination since aneurysms became important clinically when they became large enough to exert a haemodynamic effect (Holmes and MacFadyen, 1964; Björk, 1966). Abrams *et al.* (1963) have suggested the following criteria for surgery in patients with a ventricular aneurysm: (a) patients with expansile ventricular aneurysm; (b) patients with ventricular aneurysm and progressive heart failure refractory to medical treatment, in whom coronary arteriography shows adequate circulation to the remainder of the ventricle; and (c) patients with ventricular aneurysm in whom repeated systemic emboli are not controlled with anticoagulants. Chapman *et al.* (1961) believe that excision of aneurysm and ventricular repair is rational treatment for aneurysm following myocardial infarction when the lesion is of sufficient size to impair cardiac function.

The significance of the abnormalities which we have been able to demonstrate is not yet clear. In one case we have had confirmation at necropsy. The site of 16 out of 20 of the bulges and areas of abnormal pulsation corresponded to the electrocardiographic site of the infarct. This is contrary to the experience of Kurtzman and Lofstrom (1963) who found a poor correlation between the location of the myocardial infarction electrocardiographically and the area of abnormality seen fluoroscopically. We feel that the bulges, at least, indicate aneurysm formation (Holmes and MacFadyen, 1964) but admit that a prolonged follow up will be necessary for final evaluation.

That these abnormalities are associated with myocardial infarction if not with post-infarction aneurysm is confirmed by our study of 20 control patients who gave no history of any episode suggesting myocardial infarction or ischaemia and who were undergoing barium meal examination. A pleuro-pericardial adhesion was seen in one patient, but examination of his electrocardiogram revealed Q waves in the corresponding site, suggesting that at some time he had sustained a silent infarction.

The pattern of persisting R–ST elevation after the infarct is regarded as highly suggestive of aneurysm formation (Parkinson, Bedford, and Thomson, 1938; Myers, Klein, and Hiratzka, 1948; Ford and Levine, 1951; Moyer and Hiller, 1951). If this had been our indication for further radiological investigation, features suggestive of aneurysm would have been seen in only 11 patients.

It is often stated that ventricular aneurysm is a common sequel of inadequate bed-rest in the early stages after coronary occlusion. Moyer and Hiller (1951) state that only 3 of their 20 cases of ventricular aneurysm had an adequate period of bed-rest after the infarction and feel that this strengthens the argument that early ambulation following myocardial infarction plays an important role in aneurysm formation. Dubnow *et al.* (1965), on the other hand, found that duration of bed-rest was not a factor in aneurysm formation.

We have studied this problem in our patients. We were engaged in a trial of early mobilization and can divide our patients into two groups. In one group the patients were treated with strict bed-rest for 25 days and kept in hospital for 5 weeks; in the

other group the patients were kept in bed for 14 days but allowed considerable mobility in bed and discharged home after 3 weeks. We find that there is no significant difference in the numbers of bulges or areas of abnormal pulsation between these two groups.

The necropsy studies mentioned previously (Lisa and Ring, 1932; Appelbaum and Nicolson, 1935; Parkinson *et al.*, 1938; Douglas *et al.*, 1962; Abrams *et al.*, 1963; Dubnow *et al.*, 1965) all report a high incidence of aneurysm formation which is in excess of that found in clinical studies. Aneurysm may increase mortality rate (Dubnow *et al.*, 1965) and may therefore be over-represented in post-mortem series. A similar criticism could be applied to our own observations for we have also studied a selected group of patients who have survived their myocardial infarction by three months or more. Aneurysms may have been present in some of the patients who died in the earlier stages, though 16 patients who died during the time the series was being collected had necropsies and no overt aneurysm was seen. This does not exclude the possibility that in life the infarcted area of weakened fibrosed myocardium might have shown as a bulge on fluoroscopy.

In the necropsy studies mentioned in our introduction the point has been made that only the occasional case of aneurysm had been diagnosed in life. This may be an indication of inadequate investigation in the post-infarction stage. Even in recent studies of the effects of early ambulation in 236 patients by Brummer and his colleagues (Brummer, Linko, and Kasanen, 1956; Brummer, Kallio, and Tala, 1966) only 48 had a follow-up chest *x*-ray examination six months after the infarct and only one aneurysm was detected. If we had taken only postero-anterior views of the heart in our patients, few of these abnormalities would have been detected.

Pericardial adhesions have been seen in 11 patients. These probably represent the results of healed pericardial inflammation, but in view of our experience at the necropsy described above, where the pericardium was tightly adherent to the underlying aneurysm, and in view of the number of patients (4) who showed persistent R–ST elevation 6 months after the infarct, we wonder whether some cases of aneurysm have not been concealed by overlying adhesions, as suggested by Moyer and Hiller (1951) and Holmes and MacFadyen (1964).

It is evident that more abnormalities would be defined if cinefluorograph records were made (Kurtzman and Lofstrom, 1963). We agree with those authors that such studies are valuable in the assessment of myocardial infarction and we intend to follow up this group of patients over a prolonged period in order to answer some of the problems raised in this paper.

Summary

Seventy-five male patients who had survived a myocardial infarction which occurred more than three months previously were examined by cardiac fluoroscopy. A variety of abnormalities was detected and features suggestive of aneurysm formation were seen in 27 per cent of them. The significance of these abnormalities and their relation to electrocardiographic findings are discussed.

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